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10/559,806	12/08/2005	Jin-Soo Kim	Q91925	1556

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SUGHRUE MION, PLLC
2100 PENNSYLVANIA AVENUE, N.W.
SUITE 800
WASHINGTON, DC 20037

EXAMINER	
DUNSTON, JENNIFER ANN	
ART UNIT	PAPER NUMBER
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/559,806	KIM ET AL.	
	Examiner	Art Unit	
	Jennifer Dunston	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *29 January 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-81* is/are pending in the application.

4a) Of the above claim(s) *14,15,21-25,27,28,39-48 and 53-81* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-13,16-20,26,29-38 and 49-52* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *08 December 2005* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *12/8/2005;4/14/2006*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claims 1-81 are pending in the instant application.

Election/Restrictions

Applicant's election without traverse of Group I and the zinc finger motif set QSHR-RDHT-RSHR, which is identified as F435 in Table 2, an HIV tat domain as the protein transduction domain, and a KOX repression domain as the further domain in the reply filed on 1/29/2008 is acknowledged. In the reply filed 1/29/2008, Applicant indicated that claims 1-13, 16-20, 26, 29-38 and 49-52 are readable upon the elected species.

Claims 14-15, 21-25 and 27-28 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected species, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 1/29/2008. Claims 39-48 and 53-81 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 1/29/2008.

An examination on the merits of claims 1-13, 16-20, 26, 29-38 and 49-52 follows.

Information Disclosure Statement

Receipt of information disclosure statements, filed on 12/8/2005 and 4/14/2006, is acknowledged. The signed and initialed PTO 1449s have been mailed with this action.

Claim Objections

Claim 7 is objected to because of the following informalities: the claim does not end in a period and thus is not a complete sentence. Appropriate correction is required.

Claim Rejections - 35 USC § 101

35 U.S.C. 101 reads as follows:

> Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

Claims 49-52 are rejected under 35 USC § 101 because the claimed invention is directed to non-statutory subject matter. The claimed cells are present or intended to be present in a human being, which is non-statutory subject matter (e.g., specification, paragraph bridging pages 4-5). As such, the recitation of the limitation "isolated" would be remedial. See 1077 O.G. 24, April 21, 1987.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

Claims 1-3, 8-9, 11-13, 16-20, 26, 29-34, 36, 49-50 and 52 are rejected under 35 U.S.C. 102(e) as being anticipated by Sera (US Patent Application Publication 2003/0134350 A1, cited in a prior action; see the entire reference).

Regarding claims 1, 19 and 20, Sera teaches a fusion protein comprising a plurality of zinc finger domains and a heterologous protein transduction domain for cellular uptake of the protein across a cellular membrane (e.g., paragraphs [0028], [0032]-[0033] and [0183]).

Regarding claim 2, Sera teaches that the protein transduction domain may be used in conjunction with other moieties such as regulatory domains, and regulatory domains can be associated with the zinc finger domains at any suitable position, including the C- or N-terminus (e.g., paragraphs [0183] and [0197]).

Regarding claim 3, Sera teaches the fusion protein comprising 3 to 6 zinc finger domains (e.g., paragraph [0028]).

Regarding claim 8, Sera teaches the fusion protein further comprising a nuclear localization signal (e.g., paragraphs [0032]-[0033]).

Regarding claim 9, Sera teaches the fusion protein comprising a zinc finger that is a wild type or naturally occurring zinc finger (e.g., paragraph [0110]).

Regarding claims 11-13, Sera teaches the fusion protein where the protein transduction domain is the HIV tat protein transduction domain of the amino acid sequence YGRKKRRQRRR (e.g., paragraph [0183]).

Regarding claims 16-18, Sera teaches that zinc finger transcription factors selectively regulate gene expression (e.g., paragraph [003]). Further, Sera teaches fusion proteins comprising 3-40 zinc finger domains (e.g., paragraph [0028]). The number of zinc finger

domains present in the fusion protein would be sufficient to selectively direct expression of at least one, but fewer than 0.01% of genes in a cell.

Regarding claim 26, Sera teaches that the zinc finger is an independent folding domain which uses a zinc ion to stabilize the packaging of an antiparallel β-sheet against an α-helix (e.g., paragraph [003]). Thus, the zinc finger domains of the fusion protein of Sera are each bound to a zinc atom.

Regarding claim 29, Sera teaches the fusion protein further comprising a transcription repression domain (e.g., paragraphs [0032]-[0033], [0125], and [0183]).

Regarding claim 30, Sera teaches the fusion protein further comprising a transcription repression domain, where the transcription repression domain is a KRAB repression domain from a human KOX-I protein (e.g., paragraph [0200]).

Regarding claim 31, the fusion protein of Sera comprising a protein transduction domain such as the HIV tat protein transduction domain (YGRKKRRQRRR) would be capable of being transduced into at least 50% of cultured HEK 293 cells in an assay in which the cells are at 3 x 10^5 cells/ml and the protein is present in the extracellular medium at a concentration of 100 micrograms/ml, because Sera teaches that the HIV Tat protein transduction domain is a cellular uptake signal (e.g., paragraph [0183]).

Regarding claim 32, the fusion protein of Sera et al would be stable for at least 0.5 hours in human tissue culture cells. The specification discloses that proteins are stable under these conditions when it is sufficiently pure (e.g., page 4, lines 14-17). Sera teaches the purification of zinc finger fusion proteins (e.g., paragraphs [0205], [0241], [0264] and [0301]).

Regarding claim 33, Sera et al teach compositions comprising the fusion protein and a pharmaceutically acceptable carrier, excipient or stabilizer (e.g., paragraph [0256]).

Regarding claim 34, Sera teaches the addition of stabilizers that modulate disulfide bond formation through thio-disulfide bond interchange (e.g., paragraph [0260]).

Regarding claim 36, Sera teaches sustained release formulations containing stabilizers that decrease disulfide bond formation, allowing release over 100 days, which would be capable of being stable in cell culture media for at least 12 hours when the composition is combined with cell culture media (e.g., paragraph [0260]).

Regarding claims 49 and 52, Sera teaches the delivery of the zinc finger fusion proteins of the invention to eukaryotic cells such as cells of humans, fish, chickens, cows, pigs, mice, etc. (e.g., paragraphs [0160]-[0162]). Delivery of the protein to the eukaryotic cell results in a cell that contains the exogenous zinc finger fusion polypeptide, but not a nucleic acid encoding the exogenous polypeptide. Sera teaches zinc finger fusion proteins comprising a protein transduction domain and a regulatory domain (e.g., paragraph [0183]), such that the polypeptide would be functional to regulate transcription of a selected subset of endogenous genes in the cell for at least 12 or 48 hours after introduction of the endogenous polypeptide.

Regarding claim 50, Sera teaches the fusion protein further comprising a nuclear localization signal (e.g., paragraphs [0032]-[0033]).

Claims 1-5, 8, 11-12, 16-20, 26, 29-33, 38, 49 and 52 are rejected under 35 U.S.C. 102(e) as being anticipated by Cox, III et al (US Patent No. 6,607,882 B1; see the entire reference).

Regarding claims 1, 19 and 20, Cox, III et al teach zinc finger fusion proteins comprising a plurality of zinc finger domains, and a heterologous protein transduction domain (e.g., column 8, line 22 to column 9, line 29; column 31, line 55 to column 32, line 27).

Regarding claim 2, Cox, III et al teach linking a protein transduction domain to a zinc finger protein as a fusion protein (e.g., column 32, lines 1-27 and 50-55). Linking the protein transduction domain to the zinc finger protein will result in the protein transduction domain being N-terminal or C-terminal to the plurality of zinc finger domains.

Regarding claim 3, Cox, III et al teach the fusion protein comprising three, four, five or six zinc fingers (e.g., column 5, lines 25-27).

Regarding claims 4-5, Cox, III et al et al teach the fusion protein that specifically binds to a site in the VEGF-A gene and can regulate transcription of the VEGF-A gene in a cell (e.g., column 8, lines 22-48; Examples).

Regarding claim 8, Cox, III et al teach the localization of the fusion protein to the nucleus, and teach nuclear localization sequences for this purpose (e.g., column 11, lines 42-48; column 31, lines 55-67; column 47, lines 37-40).

Regarding claims 11 and 12, Cox, III et al teach the fusion protein where the protein transduction domain comprises an HIV tat protein transduction domain (e.g., column 32, lines 14-17).

Regarding claims 16-18, Cox, III et al teach that the fusion protein can regulate at least one endogenous gene, but fewer than 0.01% of the genes, in a cell after the protein is contacted with a membrane of the cell (e.g., column 8, line 22 to column 9, line 28).

Regarding claim 26, Cox, III et al teach that zinc finger proteins comprise DNA binding domains that are stabilized by a zinc atom (e.g., paragraph bridging columns 9-10).

Regarding claim 29, Cox, III et al teach the fusion protein further comprising a transcription repression domain (e.g., column 9, lines 29-35; column 21, lines 16-36).

Regarding claim 30, Cox, III et al teach the fusion protein comprising a repression domain, where the repression domain is a KRAB repression domain from the human KOX-1 protein (e.g., column 22, lines 16-18).

Regarding claim 31, the fusion protein of Cox, III et al comprising a protein transduction domain such as the HIV tat protein transduction domain would be capable of being transduced into at least 50% of cultured HEK 293 cells in an assay in which the cells are at 3×10^5 cells/ml and the protein is present in the extracellular medium at a concentration of 100 micrograms/ml, because Sera teaches that the HIV Tat protein transduction domain is a cellular uptake signal (e.g., paragraph [0183]).

Regarding claim 32, the fusion protein of Cox, III et al would be stable for at lest 0.5 hours in human tissue culture cells. The specification discloses that proteins are stable under these conditions when it is sufficiently pure (e.g., page 4, lines 14-17). Cox, III et al teach the purification of zinc finger fusion proteins (e.g., column 18, line 35 to column 21, line 14).

Regarding claim 33, Cox, III et al teach a composition comprising the fusion protein and a pharmaceutically acceptable carrier (e.g., column 35, lines 38-48).

Regarding claim 38, Cox, III et al teach the composition comprising the fusion protein and a pharmaceutically acceptable carrier, where the fusion protein is capable of regulating VEGF-A transcription in cells of a subject (e.g., column 5, line 19 to column 6, line 3).

Regarding claims 49 and 52, Cox, III et al teach a eukaryotic cell that contains an exogenous polypeptide, but not a nucleic acid that encodes the polypeptide, where the polypeptide comprises a plurality of zinc finger domains and a heterologous protein transduction domain, where the polypeptide is capable of regulating transcription of a selected subset of endogenous genes in a cell for at least 12 or 48 hours after introduction of the polypeptide (e.g., column 5, line 19 to column 6, line 3; column 8, line 22 to column 9, line 29; column 31, line 55 to column 32, line 27).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 6-7, 9-10 and 50-51 are rejected under 35 U.S.C. 103(a) as being unpatentable over Cox, III et al (US Patent No. 6,607,882 B1; see the entire reference) in view of Tischer et al

(The Journal of Biological Chemistry, Vol. 266, No. 18, pages 11947-11954, June 1991; see the entire reference) in view of Jin-Soo et al (WO 2001/60970 A2; see the entire reference).

The teachings of Cox, III et al are described above and applied as before. Further, Cox, III et al teach that the exact positioning relative to the promoter, orientation, and within limits, distance, do not matter greatly for expression modulation by a zinc finger protein (e.g., column 8, lines 49-54). Cox, III et al teach that this feature allows considerable flexibility in choosing sites for constructing artificial transcription factors (e.g., column 8, lines 53-55).

Cox, III et al do not teach a zinc finger/protein transduction domain fusion protein comprising the motifs mQSHR-mRDHT-mRSHR or comprising a naturally occurring zinc finger domain obtained from a human zinc finger protein.

Tischer et al teach the sequence of the human VEGF-A promoter (e.g., Figure 2).

Jin-Soo et al teach that designed proteins can be produced in a subject cell or organism in order to regulate an endogenous gene (e.g., page 34, lines 4-11). Jin-Soo et al teach that the disclosed invention provides multiple benefits, including (i) the ability to select a DNA binding domain that recognizes a particular sequence, which permits the design of novel polypeptides that bind to a specific site on a DNA; (ii) diverse sequences are recognized by the disclosed zinc finger domains; and (iii) the structure of naturally occurring zinc finger proteins is modular such that high affinity binding is achieved by the cooperative effect of having multiple zinc finger modules in the same polypeptide chain (e.g., page 11, lines 8-25). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 39, which is a human zinc finger protein of the motif QSHR (e.g., page 54, lines 1-12). Jin-Soo et al teach that the QSHR2 zinc finger domain of SEQ ID NO: 39 binds to the DNA site containing the sequence GGA (e.g., page 54, lines 1-12). Jin-Soo et al

teach that the QSHR zinc finger can be used as a module to construct a chimeric DNA binding protein comprising multiple zinc finger domains for the purpose of recognizing a DNA site comprising the sequence GAA (e.g., page 54, lines 10-12). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 51, which is a human zinc finger protein of the motif RDHT (e.g., page 67, lines 21-30). Jin-Soo et al teach that this amino acid sequence binds the target sequence TGG, AGG, CGG, or GGG (e.g., page 67, lines 21-30). Jin-Soo et al teach that the RDHT zinc finger can be used as a module to construct a chimeric binding protein comprising multiple zinc finger domains for the purpose of recognizing a sequence comprising TGG, AGG, CGG, or GGG (e.g., page 68, lines 1-3). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 65, which is a human zinc finger protein of the motif RSHR (e.g., page 59, lines 15-25). Jin-Soo et al teach that this amino acid sequence binds the target sequence GGG (e.g., page 59, lines 15-25). Jin-Soo et al teach that the RSHR zinc finger can be used as a module to construct a chimeric DNA binding protein comprising multiple zinc finger domains for the purpose of recognizing a site containing the sequence GGG (e.g., page 59, lines 23-25).

Cox, III et al teach it is within the general skill of the art to design fusion proteins comprising a plurality of zinc finger domains and a protein transduction domain (PTD), where the zinc finger domains bind to a site in the VEGF-A gene. Tischer et al teach that the VEGF-A promoter sequence was known in the art. Cox, III et al do not teach combining naturally occurring human zinc finger proteins to bind to a site in the VEGF-A promoter; however, Jin-Soo et al teach it is within the skill of the art to combine known human zinc finger domains to bind to an endogenous gene sequence. Further, Jin-Soo et al teach specific zinc finger domains that recognize a contiguous sequence (GGATGGGGG) in the human VEGF-A promoter, as

shown by Tischer et al. Combining the domains of Jin-Soo et al to bind the VEGF-A promoter sequence disclosed by Tischer et al allows one to produce a zinc finger-PTD fusion protein capable of binding to the a genomic region preferred by Cox, III et al, where the three fingers have the motifs mQSHR-mRDHT-mRSHR. Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the zinc finger domains recognizing VEGF-A of Cox, III et al with the human zinc finger domains taught by Jin-Soo et al to achieve the predictable result of providing a zinc finger fusion polypeptide capable of binding to the VEGF-A promoter.

Claims 35 and 37 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sera (US Patent Application Publication 2003/0134350 A1, cited in a prior action; see the entire reference).

The teachings of Sera are described above and applied as before.

Sera doesn't specifically teach a composition comprising the zinc finger-protein transduction domain fusion protein, DTT, and 1-500 micromolar zinc chloride.

Sera teaches the purification zinc finger polypeptides from a solution comprising dithiothreitol (DTT) and 100 micromolar zinc chloride (e.g., paragraph [0264]).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to modify the fusion protein composition of Sera to include DTT and 100 micromolar zinc chloride. It would have been obvious to one of ordinary skill in the art at the time the invention was made to use DTT as the stabilizer that modulates disulfide bond formation through thio-disulfide bond interchange as taught in paragraph [0260] of Sera, because DTT is a thio-

containing compound. Further, it would have been obvious to one of ordinary skill in the art to include 100 micromolar zinc chloride in the composition comprising the fusion protein taught by Sera, because Sera teaches that zinc finger domains are stabilized by zinc atoms (e.g., paragraph [003]).

One would have been motivated to make such a modification in order to receive the expected benefit of providing a stable composition of the zinc finger protein-protein transduction domain fusion, because Sera teaches that zinc finger polypeptides are stable in the presence of DTT and zinc chloride in that they can be recovered at greater than 95% from a solution containing DTT and zinc chloride (e.g., paragraph [0264]). Based upon the teachings of the cited references, the high skill of one of ordinary skill in the art, and absent any evidence to the contrary, there would have been a reasonable expectation of success to result in the claimed invention.

Claims 10 and 51 are rejected under 35 U.S.C. 103(a) as being unpatentable over by Sera (US Patent Application Publication 2003/0134350 A1, cited in a prior action; see the entire reference) in view of Jin-Soo et al (WO 2001/60970 A2; see the entire reference).

The teachings of Sera are described above and applied as before.

Sera does not teach the fusion polypeptide where the wild type zinc finger domains are naturally occurring human zinc finger protein domains.

Jin-Soo et al teach that designed proteins can be produced in a subject cell or organism in order to regulate an endogenous gene (e.g., page 34, lines 4-11). Jin-Soo et al teach that the disclosed invention provides multiple benefits, including (i) the ability to select a DNA binding

domain that recognizes a particular sequence, which permits the design of novel polypeptides that bind to a specific site on a DNA; (ii) diverse sequences are recognized by the disclosed zinc finger domains; and (iii) the structure of naturally occurring zinc finger proteins is modular such that high affinity binding is achieved by the cooperative effect of having multiple zinc finger modules in the same polypeptide chain (e.g., page 11, lines 8-25). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 39, which is a human zinc finger protein of the motif QSHR (e.g., page 54, lines 1-12). Jin-Soo et al teach that the QSHR2 zinc finger domain of SEQ ID NO: 39 binds to the DNA site containing the sequence GGA (e.g., page 54, lines 1-12). Jin-Soo et al teach that the QSHR zinc finger can be used as a module to construct a chimeric DNA binding protein comprising multiple zinc finger domains for the purpose of recognizing a DNA site comprising the sequence GAA (e.g., page 54, lines 10-12). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 51, which is a human zinc finger protein of the motif RDHT (e.g., page 67, lines 21-30). Jin-Soo et al teach that this amino acid sequence binds the target sequence TGG, AGG, CGG, or GGG (e.g., page 67, lines 21-30). Jin-Soo et al teach that the RDHT zinc finger can be used as a module to construct a chimeric binding protein comprising multiple zinc finger domains for the purpose of recognizing a sequence comprising TGG, AGG, CGG, or GGG (e.g., page 68, lines 1-3). Jin-Soo et al teach the amino acid sequence of SEQ ID NO: 65, which is a human zinc finger protein of the motif RSHR (e.g., page 59, lines 15-25). Jin-Soo et al teach that this amino acid sequence binds the target sequence GGG (e.g., page 59, lines 15-25). Jin-Soo et al teach that the RSHR zinc finger can be used as a module to construct a chimeric DNA binding protein comprising multiple zinc finger domains for the purpose of recognizing a site containing the sequence GGG (e.g., page 59, lines 23-25).

Because Sera teaches fusion polypeptide comprising wild type zinc finger domains and a protein transduction domain, and Jin-Soo et al teach naturally occurring human zinc finger domains for the modular design of zinc finger transcription factors, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute one or more zinc finger domains of Sera with one or more zinc finger domains of Jin-Soo et al in order to achieve the predictable result of providing a zinc finger fusion polypeptide capable of binding to the sequence recognized by the cooperative binding of each zinc finger domain.

Conclusion

No claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Jennifer Dunston whose telephone number is 571-272-2916. The examiner can normally be reached on M-F, 9 am to 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Joseph Woitach can be reached at 571-272-0739. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would

like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Jennifer Dunston, Ph.D.
Examiner
Art Unit 1636

/JD/

/Celine X Qian Ph.D./
Primary Examiner, Art Unit 1636